April, 1952

CLINICAL MEDICINE

for the family physician

Wallace Marshall, M.D.

TABLE *of* CONTENTS

59th year of publication

Vol. 59, No. 4 April, 1952

EDITORIAL

Man in Numbers, II 157
by Frederic R. Stearns, M.D., Editor

ORIGINAL ARTICLES

The Changing Aspect of Middle Ear and Mastoid Disease . 160
by John H. Barrett, M.D.

Myths, Misconceptions and Mismanagement of Salivary Tumors . 165
by C. E. Nelson, M.D.
and Paul H. Deeb, M.D.

The Treatment of Acne 169
by Everett C. Fox, M.D.

Trigger Fingers and Thumbs 171
by E. E. Sprecher, M.D.

Coxsackie Viruses in Human Disease 173
by Edward C. Curnen, M.D.

Relationship of Pyelonephritis and Hypertension and its Prognostic Outlook 175
by E. J. Tanquist, M.D.

CASE PRESENTATIONS, Conversion Hysteria 182
BOOK REVIEWS 183
DIAGNOSTIC SUGGESTIONS 186
THERAPEUTIC SUGGESTIONS 187
NEW PHARMACEUTICAL PRODUCTS 188
SEND FOR THIS LITERATURE 189

WALLACE MARSHALL, M.D., of Two Rivers, Wisconsin, graduated from Northwestern University Medical School, 1932. During his internship at Wesley Hospital, Wichita, Kansas, he founded the Department of Speech Pathology (Institute of Logopedics). Formerly Instructor of Physiological Chemistry and Medicine at the University of Alabama Medical School and Associate Professor of Physiological Chemistry at Spring Hill College, Mobile, Ala. In 1940 he isolated the vasoconstrictor substance 'Kutapressin' from the liver. He is now a general practitioner in Two Rivers, Wisconsin, professorial lecturer in Scientific Research at St. Norbert College, West De Pere, Wisconsin, Member of the Board of Directors, American Medical Writer Association, Charter Member of the American Federation for Clinical Research, Member of the Committee on Education of the Wisconsin Academy of General Practice and Associate Editor of Medical Times.

Man In Numbers, II

FREDERIC R. STEARNS, M.D., *Editor*

Some time ago we outlined here, at random, a few examples of quantitative aspects of man. It appeared that this numerical breaking up of an organic entity met with some interest among our readers so that we have been encouraged to enlarge upon this subject again. As we have stated in the previous editorial, it is obvious that all attempts at quantitative views in biology in general and in human biology in particular are disruptions of a qualitative integration and continuity. Yet, exact science is based essentially on quantitative values; therefore, any quantification of a properly inseparable whole gratifies the analytic inclination of the natural scientist.

Pondering first on some facts refering to the propagation of the human race, one may be impressed by the statement that only 40% of American males are highly fertile during their productive years, that 30% to 40%, though fertile, will, without medical advice, have less children than they desire, and that about 20% to 25% will probably never become fathers even with medical care. As it is known, the human egg is about 1/250 to 1/175 of an inch in diameter —this is 250,000 times as large as a spermatozoon—and weighs about 1/20,000,000 of an ounce. A single semen discharge may contain as many as 225 million sperm cells, 75,000,000 to 150,000,000 per cc. In practical experience 80 million moving spermatozoa are required in the entire ejaculate for conception. After conception the intra-uterine growth rate of the fetus, as determined by the volume of the fetus, is more than 10,000 in size during the first month of life; in succeeding months the relative monthly growth rate may be expressed by the figures: 14; 11; 1.75; 0.82; 0.67; 0.50; 0.47; and 0.45. In view of this decreasing growth rate of the fetus with increasing organismic integration, it may be interesting to compare the growth potentiality of a single cell, experimentally. Woodruff in 1911 has shown in experiments with paramecium that a single cell, originally isolated, "possessed the potentiality to produce similar cells to the number represented by 2 raised to the 3039th power, or a volume of protoplasma approximately equal to 10^{1000} times the volume of the earth."

It may be stimulating to muse on the production of secretions of the human body in quantitative respect. Nasal secretion, for instance, amounts within 24 hours in excess to one liter. In this connection it may be mentioned that physiologists have calculated that the amount of moisture necessary, on the basis of a 24 hour supply of 21 cubic feet of air for respiration within the nose, may aggregate to 960 cc. The fasting stomach of man secretes 8 to 10 cc. of gastric juice per hour, or 190 to 240 cc. per day in reality, the quantities are much larger in the nonfasting individual. A normal adult person secretes 15 cc. of bile per kilogram of body weight every 24 hours, or about 1,000 to 1,1000 cc. of bile in a man weighing 60 to 73 kilogram (others have given the 24 hour quantity of bile secretion only as between 500 and 800 cc.). The secretion of pancreatic juice amounts to 500 to 800 cc. daily. Saliva is secreted in a quantity of approximately 15 cc. per hour; thus, the 24 hour amount aver-

ages from 600 to 1,500 cc. The total outflow of lymph through the thoracic duct has been estimated to equal between 50 to 120 cc. per hour, or 1,200 to 2,880 cc. per day. The total amount of cerebrospinal fluid has been calculated as about 150 cc.; this is the quantity of fluid which is present at a given time. Yet, cerebrospinal fluid is continually reabsorbed and formed so that much larger quantities would be collected if a canula would be placed in the subarachnoid space. Regards sweating we distinguish between two kinds of sweat glands. We are considering here only the eccrine glands which are by far in the majority and which are distributed over the whole body. There are from two to three million of these glands in the skin; placed end to end, they would extend over 2 miles. Sweat secretion may be subdivided into insensible perspiration and true sweating. About 600 to 800 grams of water are evaporated every 24 hours by insensible perspiration. As to true sweating, maximum rates of 1 to 2 liters per hour have been observed; heavy labor in hot, dry weather may cause secretion of 8 to 11 liters of sweat in 5 to 8 hours of activity. In this connection it may be pointed out that, contrary to common opinion, the liquid that the average American takes on in greatest quantities, is plain water. He consumes somewhat more than a quart a day, including the water in the food. Thus, he drinks a total of about 6,000 gallons, three quarters of a tank car, of water during an average life span of 64 years.

Broaching briefly the subject of the endocrine glands, we just give two examples. In a time in which diabetes mellitus has become statistically so much into the foreground of morbidity calculations, it may be worth-while knowing that the normal pancreas has approximately two units of insulin per gram. The number of islands of Langerhans in the pancreas vary between one fourth of a million to one and three fourth million. Recent experiments have shown that the pancreas secretes 30 to 40 units of insulin daily for a fasting man weighing 70 kilogram. As to the iodine in the thyroid gland, it is present in quantities of about a quarter of a grain (15 mg.). Three and a half times its weight of blood passes through the thyroid gland each minute; yet, in the blood there is only a total of 5 to 6 micrograms of iodine per 100 cc., or a concentration of thyroxin or dijodothyrosine of the order of 0.0001 per cent.

A few remarks on the nervous system may again be included. The surface extent of the forebrain of the fish is 19% of the whole brain; in the rabbit, 68%; in the dog, 75.5%; in the gorilla, 87%; in the human child two years of age, 90%. The human brain at birth weighs 350 grams. The weight doubles within 9 months and the weight at age 15 averages 1,350 grams. It is of anthropological significance that the brain weights of various races do not differ considerably. Average brain weights are as follows: in the Caucasian race, male, 1350 Gm., female, 1216 Gm.; in the African race, male, 1293 Gm., female, 1183 Gm.; in the Chinese race. male, 1357 Gm., female, 1239 Gm.; and in the Asiatic race as a whole, male, 1304 Gm., female, 1194 Gm. We have pointed to the number of neuronal connections in the brain in a previous editorial; we may add here that about 20,000 neuron units are situated under one square mm. of cortex. Considering touch sensation, it is known that there are spots for cold (Krause's end bulbs) and for warm (Ruffini's corpuscles) sensation in the skin. The former are more numerous than the latter. It has been established that there are 250,000 cold spots and 30,000 warm spots distributed over the cutaneous surface. With respect to hearing, the fibers of the cochlear branch of the auditory nerve arise

in nerve cells of the spiral ganglion situated in the cochlea; each ganglion cell sends a fiber to the brain in the VIII nerve and another fiber ending around the sensory hair cells of the organ of Corti. There are, in this organ, at least 3,500 inner hair cells and 13,000 outer hair cells, a total of 16,500 hair cells.

There are 696 skeletal muscles with striped fibers in the body; they account for 42% of its weight in men and 36% of its weight in women. The muscle fibers vary in length from 1/25 inch to 2 inches; it takes 600 of them, put side by side, to cover an inch. Each fiber is transversely striped and contains 8,000 stripes to the inch. The absolute power of the human muscle of one square centimeter area is estimated by Hermann as 6.24 kilogram. Absolute power is calculated according to the formula $W=L \times H$. W is the work performed, L is the load and H is the lift. When the load is just sufficient to counteract the contraction of the muscle, no work has been done, and H is zero. This is the absolute power of the muscle according to Weber.

As to measurements of special organs, it has been long recognized that the skin is the largest organ of the human body; it comprises about 16% of its total weight. The area of the human skin is about 20 square feet; its volume is 2,400 to 2,800 cc. and its weight between 3,000 and 3,500 Gm.

In the new-born child the stomach is hardly as large as a hen's egg and its capacity is about 1 oz. (28cc.) In the adult, in a fully distended condition, the length is 10 to 11 inches and the greatest diameter 4 to 4½ inches; the capacity is about 40 ounces (one quart). Measurements of the small intestine in 100 bodies revealed an average length of 22 feet; the longest was 31′ 6″ and the shortest 15′ 6″, a difference of 16 feet, or more than 100% of the minimum length. There are, however, nearly 900 circular folds in the small intestine which more than double the surface. Furthermore, there are several million of villi (in the duodenum about 20,000 per square inch) which increase the absorbing surface of the small intestine to about 8,800 square inches (60 square feet) which without villi would only be 1,700 square inches. The lungs weigh more than 2 lbs. The respiration surface is between 700 and 900 square feet which takes care of 20 cubic inches of air with each respiration or 300 cubic feet of air every minute.

We shall conclude with a rather sad numericical statement, published by the American Dental Association, namely, that the average American who reaches the age of 60 has only 2 of his 32 permanent teeth left.

NOTES on MEDICAL STATISTICS

PERFORATED PEPTIC ULCER

"The average operative mortality in 147 surgically treated cases of proved perforated gastroduodenal ulcers, as reported by 13 authors, was 5.2 per cent . . . This operative mortality is less than the 6.4 per cent obtained by the proponents of the medical treatment of perforated peptic ulcers." (D. C. Collins. Med. Woman's J. 3:14, May-June 1951—reprinted from Internat. Rec. of Med. June 1951).

The Changing Aspect of Middle Ear and Mastoid Disease

JOHN H. BARRETT, M.D.

Houston, Texas

What would be my conception of middle ear and mastoid infections if I were doing general practice today? Having been a general practitioner for six years before specializing, stimulates my interest in this question. Since the advent of chemotherapy and the antibiotics, both the symptoms and treatment of acute otitis media and mastoiditis have been altered. If I were not limiting my practice to otolaryngology, what would be my ideas on this subject? Would I still consider it as taught twenty years ago, would I say it is one of the passing diseases which is only of academic interest, or would I know it as taught to the medical student of today? In presenting this subject, I would like to consider the questions which I would ask and in my discussion attempt to answer them. To shorten the presentation, only typical acute and chronic infections of the middle ear and mastoid will be considered.

Have the sulfonamides and the antibiotics eliminated acute otitis media and its complications?

In the vast majority of cases acute otitis media is the result of extension of some disease process in the nose or throat. The eustachian tube presents an avenue for spread of the infection into the middle ear. The common cold and acute infections of the nasopharynx and pharynx, especially of the adenoids and tonsils, have always been the most frequent etiological offenders. Among the other diseases frequently accompanied by otitis media are: pneumonia, influenza, sinusitis, and the acute infectious diseases such as scarlet fever, pertussis, measles, diphtheria, and mumps. Preventative medicine, as well as the introduction of new drugs, has helped to eliminate or lessen the severity of these infections. Some of these are now among the vanishing diseases which seldom occur and, when they do, are cured or controlled before complications develop. Bacteriologically purulent otitis media is usually due to one of the streptococci, staphylococci, pneumococci, or influenza bacilli. Most of the organisms of this group are sensitive to one or more of the new drugs. On first thought it might appear that otitis media is also passing from the scene. More careful consideration makes one realize that the disease is still frequent and in some instances is increasing. Colds, measles, mumps, sinusitis, and infection of the adenoids and tonsils still occur. Extension into the middle ear may be a complication of any of them. Air travel with its resulting areo-otitis, the increasing use of nasal feeding and intubation tubes, injuries to the ear in traffic accidents, and the popularization of the swimming pool have actually increased the predisposition to middle ear infection.

Has the symptomatology and treatment of acute middle ear and mastoid infections been changed by the introduction of new drugs?

The symptoms and signs of acute otitis media are classical. There is usually a history of some preceding acute upper respiratory infection followed by pain in the ear, fever, leukocytosis, and an acutely inflamed tympanic membrane. In some of the

less virulent infections, only catarrhal otitis media develops and the drum shows vascular congestion instead of an acute inflammation. The pain is usually worse at night. Most untreated cases go on to suppuration with spontaneous rupture of the tympanic membrane, followed by purulent drainage. In hypovirulent infections, the pathological process may resolve with absorption of any exudate formed in the middle ear. After drainage is established, the disease may subside or it may extend through the aditus ad antrum and cause an acute mastoiditis. The symptoms of acute infection of the mastoid are: fever, pain and tenderness over the mastoid, drainage from the tympanum, sagging of the posterior-superior canal wall, leukocytosis, and x-ray evidence of involvement of the mastoid cells. Meningitis, labyrinthitis, brain abscess, lateral sinus thrombosis, or subperiosteal abscess may occur as a complication.

Before the introduction of sulfanilamide the treatment of acute otitis media and its complications consisted of surgical drainage of the middle ear, supportive symptomatic treatment, and mastoidectomy when this part of temporal bone became involved. Early myringotomy offered the best prognosis for the infection to subside without complications. When mastoiditis occurred, a simple mastoidectomy was indicated. The term simple is a misnomer as the operation should be designated as a complete mastoidectomy. If the operation was not performed, one must pray for resolution of the infection, suffer the complications of inner ear or intracranial extension, or expect a chronic tympanomastoiditis. As far as the surgical procedure is concerned, very few improvements have been made since this operation was described by Whiting[1] in 1905. The use of electrically driven burrs instead of curettes has been advocated and Lempert[2] devised an endaural approach to replace the post-auricular incision. The removal of all infected bone from the mastoid was so thoroughly described by Whiting that it left little for the future surgeons to perfect as far as the complete mastiodectomy was concerned. The difficulty has been the surgical skill required to remove all the pathology and avoid injury to the adjacent anatomy.

Soon after the introduction of sulfanilamide, the miraculous response of middle ear infections to this drug and its associated compounds was observed by numerous clinicians including: Fenton[3], Galloway[4], Babcock[5], Bowers[6], Livingston[7], Cunning and Guerry[8], and Rubin and associates[9]. It was found that sulfonamides would cure a large percentage of these infections and their complications, if the organism was sensitive and the drug was given in adequate amounts. The administration of these drugs causes a quiescence of the clinical signs, giving the impression that the infection has completely subsided. Maybaum and associates[10], Fenton[3], Bowers[6], Converse[12], Lillie[13], Kopetsky[14], Scal[11], Babcock[5], and Maxwell[15], observed that the familiar signs and symptoms of acute otitis media and mastoiditis are changed by chemotherapy. Many of the infections were still present after the clinical signs had subsided. When penicillin became available, its beneficial effect on ear infections was observed by many including: Struble[16], Karatay[17], and Putney[18]. This drug was found to be more effective than the sulfonamide compounds. Pulaski and Matthews[19], and Hold and Snell[20] reported the beneficial response of otitic infections to streptomycin therapy. More recently the newer drugs of the mycin group including: aureomycin, chloromycetin, and terramycin, have been found to be equally effective

in ear infections. It has been observed that all of these drugs lessen the severity of acute otitis media and its complications, and will effect a cure in the majority of cases if the therapy is adequate. The masking of symptoms by chemotherapy has been reported by Gilmore[21] and Coleman[22], and later I[23] reported similar observations in treating acute otitic infections with penicillin.

Most clinicians agree that treatment by either chemotherapy or the antibiotics alters the classical signs and symptoms of acute otitis media and mastoiditis. All of these drugs cause a quiescence of the infectious process with a reduction of the severity of symptoms. The elevation of temperature is retarded and the pain is not as severe. The tympanic membrane is not red and bulging as in untreated cases, but often appears dull and thickened, with hyperemia of the vessels along the handle of the malleus. The drum is more characteristic of catarrhal otitis media. When perforation occurs, or myringotomy is necessary, the amount of drainage is variable. It may be scant or moderate, and is often sterile on bacteriological examination. After all clinical symptoms have subsided and the drug has been discontinued, the drainage, pain and fever may recur. Recurrence of pain and fever is more frequent than aural discharge. Some few organisms are resistant to all these drugs and such cases usually have pain and profuse drainage.

The symptoms of acute mastoiditis are even more masked than those of otitis media. Pain and tenderness over the mastoid along with sagging of the posterior superior canal wall is seldom encountered. Leukocytosis is often slight or may be absent, as all of these drugs reduce the virulence of the infection and the sulfonamide compounds tend to decrease the white cells. X-ray evidence of mastoiditis persists long after the clinical symptoms have subsided. It is possible that some failures in treatment have been due to discontinuing the drug before the roentgenologic changes have returned to normal. The characteristic symptoms of complications such as lateral sinus thrombosis, meningitis, and labyrinthitis, are also altered but fortunately these complications are very rare today. Positive blood cultures are very infrequent. It must be remembered that patients may become allergic to any of these drugs and develop a marked pyrexia before other allergic symptoms have become apparent.

Since the advent of chemotherapy and the antibiotics, there has developed in the medical profession a somewhat dangerous sense of security so far as ear infections are concerned. Many physicians have advocated treating acute otitis media by chemotherapy or antibiotics without performing a myringotomy. The idea that an acutely inflamed drum need not be opened is unsound and has been responsible for complications that could have been avoided. When acute otitis media develops, better results can be obtained by a myringotomy followed by one of these drugs. There is much less chance of the organism becoming resistant to therapy when adequate drainage is present and less medication is required to effect a cure. Lederer[24] states that many physicians indiscriminately and fearlessly dose their patients without having established a diagnosis, and he accuses us of being gadgeteers of medicine because we administer these drugs in numerous methods which will not afford an adequate blood level. Scientifically, a bacteriological diagnosis should be established before either chemotherapy or antibiotics are started. From a practical standpoint, this often is not possible

Treatment should be continued for two or three days after all clinical signs have subsided, and should be reinstituted if the symptoms recur.

Acute mastoiditis will respond to either type of drug if the infection is still in the phlegmonous stage, but once it has progressed to the coalescent form with bone destruction they offer little chance of a permanent cure. If the mastoid cells are broken down and walled off pus is present, surgery is indicated. A complete mastoidectomy with removal of all the infected bone should be performed. Either the postauricular approach or the Lempert[2] endaural technique may be employed; however, it seems that the postauricular operation is still preferable for this type of infection. After all infection is removed by surgery, it should be followed by chemotherapy or better by one of the antibiotics. Since using antibiotics after surgery, it has been possible to close the postauricular incision without drainage, and the postoperative fistula has disappeared. The local use of these drugs in the mastoid cavity has been tried, but it was found not to give as good results as when an adequate blood level is maintained by oral or parenteral administration; and in some instances it caused complications. Although the number of patients requiring surgery for acute mastoiditis has been very markedly reduced, neglect or drug resistance are still responsible for a few cases.

Has chronic middle ear and mastoid infection been changed by these new drugs?

Chronic infection of the middle ear or mastoid is the sequela of an unsuccessfully treated acute infection. When chronic suppuration develops in the tympanic cavity, there is always extension into the mastoid antrum. If the infection occurs in early life, pneumatization is arrested and a sclerotic mastoid develops. If the mastoid is developed, then chronic infection is present throughout the bone. There is always a perforation of the tympanic membrane when chronic otitis media is present. The gravity of the infection may be judged by the location of the perforation. The inflammatory process may finally subside with cessation of drainage and formation of scar tissue adhesions. This is more likely to occur when the perforation is in the inferior part of the drum than when it is in Shrapnell's membrane. Serious complications are infrequent in inferior quadrant or large central perforations. When infection is present the tympanum and mastoid cavity may be filled with pus or granulations, and in some by a cholesteatoma. This is nature's attempt to line the cavity with epithelium from the margin of the perforation. A fetid otorrhea is usually present. Some impairment of hearing is present in chronic otitis media. The infection may remain quiescent with a perforation present and recurring aural drainage. An acute exacerbation of the infection may occur with extension into the inner ear, facial nerve, lateral sinus or cranium.

In the era before sulfanilamide, treatment of chronic tympano-mastoiditis consisted of local treatment of the ear, symptomatic therapy and surgery. Various drops such as boric acid in alcohol and powders such as Sulzberger's (iodine and boric acid) have been advocated. This type of treatment helped to keep the ear clean and dry, but the number of cures was minimal. If the granulations and drainage could not be controlled, or complications occurred, surgery was indicated. The complete mastoidectomy was not suitable for this type of pathology. The radical mastoidectomy was the operation of choice. The procedure consists of cleaning out the mastoid and middle ear (except for the stapes), and removal of the posterior-superior canal

wall, converting the mastoid and tympanum into one caivty. Removing the tympanic membrane and the lever mechanism of the ossicles always gave about a 20 decibel drop in hearing, however in most cases requiring operation the hearing was already below the serviceable level. When the perforation was in Shrapnell's membrane and the hearing was good, a modified radical mastoidectomy could be employed. The procedure consisted of cleaning out the mastoid cavity and removing the posterior-superior canal wall but leaving intact the tympanic membrane and ossicles. This operation removed the pathology but conserved the hearing present. When sulfonamides and antibiotics were found to be effective in acute infections they were tried for the chronic cases. Some of the early observations with sulfonamides, as Lawson's[25], were promising, but as more experience was gained it was found that these drugs were useful in controlling acute exacerbations but had little effect on the chronic infection. In cases with cholesteatoma the results were very disappointing. Later Krauss[26] reported similar observations with penicillin. The other antibiotics have not proven satisfactory for these chronic infections. Brown and Owen[27] have used glycerite of hydrogen peroxide locally with beneficial results, and Unger[28] has reported good results from using deep lavage, aeration, and application of gold foil membrane bearing scarlet red ointment. If chronic tympano-mastoiditis fails to respond to a combination of local treatment and chemotherapy or antibiotics, then a radical or modified radical mastoidectomy is indicated.

Although very little has been added to the complete mastoidectomy, remarkable advancement has been made in the radical procedures. Lempert's[29] work on the fenestration operation has popularized the endaural approach, removal of pathology with electrically driven burrs, good illumination and the use of magnification in working in the middle ear. All of these additions have helped perfect the radical mastoidectomy. The endaural operation gives better exposure of the tympanum and mastoid antrum where the majority of the pathology occurs. The use of magnification has eliminated many of the hazards such as injury to the facial nerve and incomplete removal of pathology from the tympanic cavity. All mastoid surgery can be performed through the endaural approach, however it might be difficult to expose retro-sinus cells in a well developed mastoid. Many surgeons still use the post-auricular approach with equally good results. The use of chemotherapy and especially the antibiotics in post-operative treatment has reduced the incidence of complications and hastened healing. Post-operative treatment should be continued until epithelization of the cavity is complete. The use of these new drugs has greatly reduced the length of this treatment. Skin grafting of the mastoid cavity has also been employed to hasten healing. Regardless of the approach used, any person who has had a radical mastoidectomy has a surgical defect for the remainder of his life. After healing is complete, it is necessary to clean the cavity at intervals to remove epithelial debris and avoid secondary infection. These operations are performed to remove pathology and not to improve hearing. If a bilateral operation is required, a hearing aid or a Pohlman[30] artificial ear drum may be necessary to rehabilitate the individual's hearing. Chemotherapy and antibiotics are effective in curing most acute ear infections, but in chronic tympano-mastoiditis surgery is usually necessary to elimnate the pathology.

Conclusions

1. The introduction of chemotherapy and antibiotics has so changed our conception of acute and chronic infections of the middle ear and mastoid that we should reconsider exactly how it has changed the symptomatology and treatment of these infections.

2. Preventative medicine and the introduction of new drugs has reduced the incidence of acute otitis media and mastoiditis, but the disease still occurs and in a few instances the predisposition to this infection has increased.

3. Before the introduction of sulfanilamide the signs and symptoms of acute otitis media and mastoiditis were classical and the treatment consisted of surgical drainage of the tympanum, symptomatic therapy, and surgery for the mastoid complications.

4. After the advent of sulfanilamide it was observed that acute otitic infections responded favorably to the sulfonamide compounds.

5. Later the antibiotics were found to give better results than the sulfonamides.

6. Both chemotherapy and the antibiotics were observed to mask the symptoms of acute otitis media and mastoiditis.

7. As long as infections of the mastoid remain in the phlegmonous state, response to both of these drugs is satisfactory, but when the bone destruction occurs, the chance of cure is greatly reduced and surgery is usually necessary.

8. In the past, the treatment of chronic tympano-mastoditis was local treatment of the ear or surgery.

9. Sulfonamides and antibiotics were found to aid in controlling acute exacerbations of chronic infections, but offered little chance of a permanent cure.

10. When symptoms of chronic tympano-mastoiditis persist, the radical mastoidectomy is the treatment of choice.

(Editorial note: references identified upon request)

Myths, Misconceptions and Mismanagement of Salivary Tumors

C. E. NELSON, M.D.
PAUL H. DEEB, M.D.

The Departments of Surgery and Radiology, White Memorial Hospital College of Medical Evangelists, Los Angeles, California

The fog is slowly being lifted as to the true nature and importance of salivary tumors. Parotid tumors in particular have been the bogey of head and neck tumors. Because of their slow growth some have taken them very lightly while others with fear and trembling have left them alone for fear that any attack upon them would mean either facial paralysis or certain recurrence. Since the benign mixed tumor has been the tumor usually thought of when tumor in the parotid gland occurs, the frequency of malignancy has often been overlooked. We feel that there are several basic causes for these difficulties and the purpose of this paper is to describe fundamental principles in management of these lesions as well as discussing misconcpetions of the problem in the past.

In the embryonic life the parotid gland has its origin in the oral cavity. It begins as a bud and grows toward the ear situated external to the facial nerve. At the point where the facial nerve divides into its two main divisions, the temporal-facial and the cervical-facial, the parotid gland butts downward in between these two filaments and behind the mandible where a smaller deep lobe is formed. The portion connecting deep lobe and main gland is called the isthmus, the external lobe being the larger and the internal lobe varying considerably in size but much smaller than the external lobe. Thus it would be seen that the facial nerve does not actually go through the substance of the parotid gland as has been taught in all textbooks of anatomy, but the gland develops around the nerve, frequently enveloping lobe fibers. By careful dissection but with small hemostatic forceps, the lobules can be dissected around the nerve, making a total parotidectomy possible without sacrificing the facial nerve. Anson and his co-workers[1] have contributed valuable information regarding the relationship of the parotid gland to the facial nerve.

Nearly three-fourths of all salivary tumors are located in the parotid gland. The remaining one-fourth are situated in the sublingual, submaxillary glands or in aberrant salivary tissue or minor salivary glands widely dispersed throughout the oral cavity. The high incidence of malignant change in these salivary tumors has been little appreciated in the past by physicians or patients. It is usually reported that about twenty per cent of all tumors of the parotid gland are malignant although in a recent survey we found that the incidence reached as high as fifty per cent[2]. In as much as the mixed tumor is the most common benign tumor of the parotid gland, and because of its slow growth, some authorities[3] recommended the attitude of leaving them alone. Others, because of the false conception of it being an encapsulated tumor have attempted to enucleate it which almost always resulted in recurrence. The soft consistency or cystic nature of certain of these tumors has led some to feel that they were benign cystic lesions whereas in fact many of them are malignant, or should be treated as such. About five per cent of parotid tumors are of benign lymphocytic type. We shall not in this discussion attempt to go into the pathology of these tumors as that subject has been amply covered by others.

Other errors in the management of parotid tumors have been the poor follow-up studies, so that recurrences were not recognized by the physician or the patient. Another factor has been the fairly frequent history of a cycle of swelling followed by considerable regression, causing the impression that the lesions were of inflammatory nature. Pain is an infrequent symptom of parotid tumors excepting in the advanced malignancies. Chronic parotitis at times constitutes a difficult problem in both diagnosis and management. History of recent origin associated with pain, low-grade rise in temperature, possible response to anti-biotics as well as the value of roentgenray examination helps to make a diagnosis. Roentgen examinations of the parotid gland should be by both the oblique views and the AP projections as well as lateral projections. The use of several angles is of value in case a stone is present and would assist in its location. Where inflammatory lesion or abscess is suspected, aspiration of contents often will show the presence of pus. Or if a stone should be present, placing needles at various angles will help to localize the stone and thus assist in its removal. The value of the use of opaque oil study in the parotid gland has been

little appreciated. The use of lipiodol in a properly anesthetized Stensen's duct orifice is of only mild discomfort to the patient when performed by experienced individuals. There is some discomfort as the duct and its branches are distended by pressure on the syringe. However, these sialograms are of value in differentiating the chronic infectious changes which produce irregularity of the gland structure deformity of the branches when visualized on the films. Should a cyst or tumor be present, the displacement of the duct system is often seen, and while diagnosis of malignancy is not well made, the actual distribution of the salivary duct radicles and their locations is of value to the surgeon who may perform the operation later. Of course, it is noted that these examinations are adjuncts to a thorough examination and history. The authors have been impressed with the frequency with which these tumors have been confused with sebacious tumors. Several of our patients give a history of having been operated upon in the doctor's office for what was thought to have been removal of a sebacious cyst.

Carcinoma of the parotid gland can occur at any age and while it is more common in the over-forty age group, we have several cases in the twenties and the youngest was sixteen years of age. One patient under observation had not reached her twentieth birthday but adenocarcinoma of the parotid gland proven by biopsy shows metastasis in both lungs on the x-ray film.

In rapidly growing tumors and in definitely proven malignancy, x-ray of bones should be carried out as they not infrequently metastasize to bone. As a precautionary measure, an x-ray of the chest should be a routine procedure.

It should be constantly kept in mind that the parotid gland is a frequent site of metastasis for tumors of skin and face and head and neck region. Lymphosarcoma frequently involves the parotid gland as well as other neck structures. In one case under the observation, Mikulicz's tumor involved the parotid gland.

Melanoma may primarily or may secondarily involve the parotid gland. One of our patients with a tumor of the parotid gland of seven years duration proved to be a melanoma without a definite primary site elsewhere.

Once the diagnosis of parotid tumor has been made we feel that its surgical removal is definitely indicated. If the tumor is known to be malignant, no attempt should be made to save the facial nerve, but should have total parotidectomy together with a radical neck resection. In tumors showing no definite signs of malignancy, if they are located in the peripheral area of the gland on the external lobe, they may be removed locally together with a liberal portion of parotid tissue on either side or an external lobectomy may be performed. If the tumor is external behind the mandible, a total parotidectomy is indicated. In the recurring mixed tumors there is danger of developing carcinoma, hence the first attack should include a wide removal of all tissue. No attempt at "enucleation" or "shelling out" should be done, in as much as this results in incomplete surgery and thus paves the way for failure of surgery and recurrence. These tumors do not have a true capsule but are surrounded by condensed parotid tissue and may have tumor tissue in this capsule-like condensed tissue substance.

Mixed parotid tumors are notor iously resistent to radiation therapy, however there are malignant tumors with cell structures that will respond, and where surgery is not possible, radiation should be used. Lymphocytic and Warthin's tumors are radiosensitive, and in these se-

lected cases a thorough course of radiation will produce good results.

Comments & Conclusions

1. Any chronic swelling of the parotid gland calls for a careful search as to the nature and cause, and if a patient does not respond well to antibiotics as well as local applications of heat and medication of the area, additional studies such as x-ray of the area, x-ray of the chest, and possible special opaque media studies would be indicated.

2. The differential diagnosis of the parotid swellings include the chronic inflammatory, the chronic granulomatous lesions, multiple benign cysts and adenomatous changes as well as neurofibroma, malignant changes of adenocarcinoma and squamous carcinoma and connective tissue malignancies such as lymphosarcoma. The simple diagnosis of mixed tumor has often been a poor camouflage of the exact nature of parotid tumors.

3. The management should include thorough surgical procedure once the diagnosis of tumor has been made. The facial nerve may be protected with careful knowledge of the anatomy and thorough surgical procedure dissecting this nerve out if the lesion is benign. However, the justification of sacrifice of a facial nerve may be made if the nature of malignancy is proven by pathological study.

4. Careful follow-up following surgical procedure including periodic roentgen examination of the chest is indicated.

5. Any minimal procedure such as "shelling out" the tumor as a minor surgical procedure is to be thoroughly condemned.

6. The fallacy that most of these lesions are innocent should be corrected.

7. A closer cooperation between the referring physician, the pathologist and the surgeon should be made in view of the fact that this lesion is difficult to diagnose and has been a source of confusion and poor management in past years.

8. Consider the possibility of parotid tumor in the under-age group.

9. Mere palpation and clinical observation of any swelling of the neck is insufficient diagnostic procedure and repeated observations of suspicious swellings should be made at the physician's office until the lesion disappears.

BIBLIOGRAPHY

1. McCormack, L. J.; Cauldwell, E. W.; and Anson, B. J.: Surgical anatomy of the facial nerve with special reference to the parotid gland, Surg., Gynec. & Obst. 80: 620-630, June 1945.
2. Nelson, Clarence E.; and Deeb, Paul H.: Recent trends in the management of parotid tumors. A report of 100 cases. Annals of Western Medicine and Surgery, V. 4, No. 11, 683-685.
3. McFarland, J. Mysterious mixed tumors of the salivary glands. Surg., Gynec. & Obst. 76:23-34. January 1943.

SIDE-GLANCES at HISTORY OF MEDICINE

OSTEITIS FIBROSA CYSTICA

was first described by F. D. von Reckinghausen. Die fibroese oder deformierende Osteitis, die Osteomalacie und die osteoplastische Carcinose in ihren gegenseitigen Beziehungen. Festschrift Rudolf Virchow, Berlin 1891, pp. 1-89. Fifty two years later the first removal of a parathyroid tumor was performed, found in relation to the disease, with good results, by F. Mandl. Therapeutischer Versuch bei osteitis fibrosa generalisata mittels Extirpation eines Epithelkoerperchens. Wien. klin. Wchnschr. 38:1343, 1925.

The Treatment of Acne

EVERETT C. FOX, M.D.

From the Department of Dermatology and Syphilology, Southwestern Medical School of the University of Texas, Dallas, Texas

Acne Vulgaris is not an insignificant or negligible disease.[1] It will in a few cases, leave disfigurement, as a result of pitting and scarring. The only way to prevent disfigurement is adequate treatment of the acne process at its inception. Acne often produces feelings of inferiority with psychologic and emotional damage which may be permanent. Acne is a disease not to be taken lightly, and it is the physician's duty to prescribe and direct the most effective treatment available.

Acne is primarily a physiologic overactivity of the pilosebaceous system which is manifest as an oversecretion of sebum by the oil glands with follicular cornification and plugging, seen predominately in adolescence.

The primary lesion of acne vulgaris is the comedone associated with the follicular hyperkeratosis. The inflammatory papule may proceed to a pustular lesion, a deep nodule or a cystic lesion with resultant scarring. Infection plays a very little part in the development of acne, and that only secondarily. The bacerial flora of the skin in patients with acne is the same as that of the skin in patients without acne and in similar numbers.

The clinical variations of the disease represent individual reactions to the basic cause and may be changed or aggravated by many factors. Heredity, glandular activity of puberty, endocrine disturbances, shift in androgen-estrogen ratio, intercurrent and focal infections, parasympathetic nervous hyperactivity, anemia, use of iodides or bromides, disturbance of water balance, digestive and intestinal dysfunctions and especially excessive fat ingestion may be of etiologic significance.

Hormonal agents account for the growth of the pubic and axillary hair, the growth of lanugo hairs, the development of apocrine glands, the stimuli to the pilosebaceous structures and the appearance of acne. Bloch observed the relationship between the occurence of comedones, papules, pustules and the appearance of such secondary sexual characteristics as axillary and pubic hair and puberty. He found that sixty per cent of girls and seventy per cent of boys had some degree of acne between the ages of six and eighteen years and that by the eighteenth year more than ninety percent were affected. The severe cases were more frequent among boys.

Recent observation of the development of acne following the administration of ACTH and Cortisone have further emphasized the importance of the steroid hormones. Hamilton showed that eunuchoid individuals do not develop acne vulgaris.

At present time there is no doubt that estrogenic substances are of benefit in many cases of acne vulgaris in females. The dose of estrogens must be adjusted individually.[2] There is no regularity of response and estrogens may even aggravate some cases of acne. Many cases of acne improve during pregnancy.

There is a relationship between hormones and Vitamin A, both are of value in acne and large doses of Vitamin A have been useful in premenstrual tension and mastodynia.

The sulfonamides and antibiotics are frequently useful agents in the control of pustular, nodular and cystic cases of acne. Sulfadiazine, sulfapyridine, aureomycin or terramycin cured many cases of severe nodular and cystic acne in Andrews' series, without other measures.[3]

Treatment must be varied depending on the many factors mentioned. A detailed history of occurence, exacerbation, relation to foods, infections, rest and to menses is useful in selection of appropriate therapy.

The treatment of acne vulgaris was recently reviewed by Sulzberger and Baer[4] and as this review indicates, the modern therapeutic approach must be selected and fitted to the individual patient. The formation of comedones must be counteracted and the removal of follicular plugs facilitated by local cleansing with soap and water and the application of drying and peeling local measures adjusted to the individual patient's skin. The excretion of sebum, irritants and allergens must be controlled by diet and elimination of culpable drugs and by the removal of foci of infection. The secondary infections may be combated by local and systemic chemotherapy, antibiotics and antiseptics. The cystic and chronic lesions are aided by evacuation, drainage, hot compresses and local antiseptics. The overactivity of the sebaceous glands, the follicular plugging due to faulty keratinization must be improved by the use of Vitamin A, irradiation by roentgen rays and ultraviolet rays, scrubbing, peeling and drying local applications and by the administration of arsenicals and hormones in severe cases.

The most important therapeutic measure is frequent cleansing with a non-greasy detergent. Avoidance of all fatty and greasy applications and elimination of greasy foods from the diet are important general measures.

The routine treatment includes:

1. Soap and water cleansing two or three times daily.
2. Supplementary cleansing of the skin with Alcohol or Alcoholic Astringents will reduce the number of comedones and the amount of infection.
3. Careful removal of comedones, and evacuation of cystic and pustular lesions.
4. Use of resorcinal and/or sulphur lotion or paste at night.
   a. Resorcinol 2-5%, Salicylic acid 2-5% in Alcohol 70%.
   b. Resorcinol, Sulphur each 2-5%, zinc oxide, Talc, glycerine aa 25.0, alcohol 70%, camphor water aa qs. 120.
   c. Many preparations of Sulphur, resorcinol combinations of lotions, creams and pastes. Acnomel, Sulfo-lac, Dermasulf, Intraderm Sulphur, Sulforcin lotion, Cream or Base.
5. A low fat diet (eliminate chocolate, iodides, bromides)
6. Weekly shampoo

Special measures in addition to above routine for severe cases.

1. Vitamin A, 50,000 units one to three times daily (Aqueous)
2. Thyroid for lipoid dysfunction. (Sutton)
3. Tuberculin, intradermally. (Van Studdiford)
4. The Sulfonamides, Antibiotics (Andrews) and Arsenic in severe nodular and cystic acne.
5. Estrogens, diethylstilbestral 0.5 mgm or Natural estrogen 0.625 mgm. daily, when other measures are ineffective for the female patient over seventeen years of age. (Way, Andrews, Sulzberger)
6. Roentgen therapy, 50 to 75 r. once weekly for not more than ten to twelve treatments for cases of severe disease with deep papules, pustules and cystic lesions when there is unsatisfactory response to other measures, but preferably not until patient is 17.

(Editorial note: references identified upon request)

Trigger Fingers and Thumbs

E. E. SPRECHER, M.D.
Seattle, Washington

The term Trigger Finger is usually applied to a condition of the fingers which causes snapping when the finger is extended or flexed. Motion of the proximal interphalangeal joint is the joint which produces the symptoms more readily. If the finger is in the extended position, the joint can be flexed about 45 degrees; a snapping and jerking then occurs before further flexion is possible. After this the joint can be freely put through a complete range of motion. With the joint in the flexed position, it can be freely extended to about 135 degrees. The snapping then occurs and the rest of the range of motion is free. A small mass is palpable in the palmar surface of the hand at about the level of the distal flexor crease of the palm. When the involved finger is moved, the mass moves with the tendon. Very little pain is associated with this condition, although there is some tenderness to palpation over the mass. If the condition becomes severe enough the finger is locked. From the flexed position, motion is free to about 135 degrees, and no further extension is then possible. The ring finger is more commonly involved. It may however, be in other fingers and very rarely occurs in more than one finger of the same hand.

This condition is caused by a nodule on the flexor tendon sliding back and forth through a constricted portion of the tendon sheath. The tendon sheaths are reinforced just proximal to the heads of the metacarpal bones by a cruciate ligament. This ligament is attached to the metacarpal neck and acts as a pulley to prevent the tendon from spanning across the joint when in a flexed position. In Trigger Fingers, this pulley becomes excessively thick and is contracted to constrict the tendon sheath. There is a fusiform enlargement of the tendon. The gross appearance of the nodule is not different than that of normal tendon tissue. Microscopic examination of the nodule reveals only tendon tissue involved with a traumatic inflammation. The condition is usually brought about by repeated minor trauma, and is seen commonly in individuals working with hand tools, which require considerable force to operate. It may also be seen in invididuals in which there is no history of undue irritation of the area. Conservative treatment consists of immobilization of the finger. By preventing the nodule from slipping back and forth through the constricted area in the sheath· the inflammation subsides and it slides through the pulley more freely. Quite often on immobilization the symptoms subside but the nodule will still be palpable. The condition usually recurs with the slightest irritation of the area· and frequently recurs without any excessive use of the hand.

Usually surgery must be resorted to, to alleviate the condition. A transverse incision is made in the distal flexion crease of the palm across the involved metacarpal. After incising the skin, dissection is carried out longitudinal to the metacarpal to avoid the risk of injuring any nerves or vessels. The tendon sheath is exposed and incised along one side until the nodule can slide freely in the tendon sheath. The sheath should be incised near the bone so that if any scarring occurs with healing, it will be away from the point of maximum friction. The

nodule is left intact and the wound closed by suturing the skin only. Postoperative immobilization is usually not necessary. This operation produces good results and if done properly there should be no loss of function or residual discomfort.

Infants develop this condition in their thumbs. The same snapping is present on motion of the interphalangeal joint. The nodule is on the flexor pollicus longus tendon. Since this tendon inserts on the distal phalanx, the snapping is caused by flexion on extention of the interphalangeal joint. The metacarpal phalangeal joint is not involved. If the condition is severe the thumb may be locked, complete extention not being possible. Many infants have their thumbs in a flexed position when they are first seen. The parents give no history of noticing any abnormality until the locking occurs. In a series of twleve cases four patients demonstrated locking at the first examination after birth. Since the flexor muscles are stronger than the extensors they are able to pull the nodule through the constricted area of the sheath when the extensors are no longer able to do so. The distal joint can be freely extended to about 150 degrees, but not beyond this. Sometimes the nodule can be pulled through the constriction passively when it can no longer be done actively. Because this condition limits extension of the distal joint, it should be recognized, to prevent unnecessary surgery from being done on this joint, which I have seen done. Occasionally a child may be seen with bilateral snapping thumbs and some have been seen with a snapping thumb in one hand and a palpable nodule in the tendon in the other thumb which produces no symptoms.

The condition differs in no way either grossly or microscopically from snapping fingers. The cause of snapping fingers is accepted to be traumatic and it is my opinion that even though this occurs in infants it is on a traumatic basis. There is nothing in the development of the embryo which would cause a congenital nodule on the tendon and there has never been any evidence of any infection in any of these cases.

Infants consistently maintain their hand with a closed fist, the thumb sharply fixed and the fingers closed tightly over the thumb. I feel that this continual marked flexion causes a kink of the tendon over the pulley, which first causes edema of the tendon, and finally the inflammation a nodule. In infants, conservative treatment is never effective. Surgery consists of incision of the sheath as in Trigger Fingers. The incision is made transversely across the base of the thumb about ⅛ of an inch proximal to the proximal flexor crease. There is practically no subcutaneous tissue directly in the flexor crease and if the incision is in the crease the resulting scar is more adherent. At this level, the nerve to the lateral side of the thumb is directly over the mid-portion of the flexor surface. The incision should be to one side or the other of the nerve, preferably to the lateral side. Care should be taken to avoid severing it. The sheath is again split along one side as near the bone as possible and nodule left intact, and the wound closed by suturing the skin only. An excellent result without recurrence is to be expected.

Coxsackie Viruses in Human Disease

EDWARD C. CURNEN, M.D.

Associate Professor of Pediatrics and Preventive Medicine, Yale University School of Medicine, New Haven, Connecticut

The Coxsackie viruses constitute a group of recently discovered filtrable agents with unusual biological properties which appear to be important as a cause of human disease. Viruses of this group were first isolated from patients with illnesses resembling poliomyelitis.[1-4] The term "Coxsackie" is derived from the name of the town in New York State where strains were first encountered by Dalldorf and Sickles.[1] Like the poliomyelitis viruses these agents have been recovered from human feces and pharyngeal swabbings as well as from sewage and flies. Experimental infections induced by them differ, however, both in respect to host range and characteristics of disease from those caused by poliomyelitis viruses.[1-3, 5, 6] Illnesses attributable to Coxsackie viruses have resembled non-paralytic poliomyelitis, epidemic myalgia or pleurodynia (Bornholm disease), herpangina ,and other acute, self-limited febrile maladies with or without distinctive features.[4, 7-12]

EXPERIMENTAL

Viruses of the Coxsackie group are peculiarly pathogenic for unweaned mice and hamsters in which they cause fatal disease frequently with paralysis.[1-3, 5, 6] In these animals susceptibility to infection diminishes rapidly after the first few days of life. These viruses have been found to induce in newborn mice degenerative lesions of skeletal muscle. In mice infected by certain strains, focal necrosis of the myocardium, generalized fat necrosis, encephalitis, and pancreatitis have also been observed.[5, 6, 13, 14] The Coxsackie viruses do not induce in monkeys paralysis and lesions typical of poliomyelitis. Information is lacking concerning the pathology of infection caused by Coxsackie viruses in man.

Viruses of the Coxsackie group are among the smallest known. They are unusually stable and are resistant to ether, penicillin, streptomycin, and chloramphenicol.[6] Viral activity is maintained at room temperature for many days. Like poliomyelitis viruses these agents withstand exposure to a wide range of pH.[15]

At least ten different immunologic types of Coxsackie virus are known to exist, and it seems probable that additional types will be encountered. Coxsackie viruses appear to be capable of causing infection in man and of stimulating a specific antibody response. It has been found that neutralizing antibodies develop early in the course of human infection with Coxsackie viruses sometimes within one week, usually within two weeks of the onset of symptoms.

CLINICAL FEATURES

The clinical manifestations of disease in patients infected by Coxsackie viruses have been noted to vary widely and have not yet been completely appraised. The variations are not merely a reflection of infection by different types. Illnesses attributable to strains of a single type (Conn. -5) have resembled in multiple patients either nonparalytic poliomyelitis or epidemic myalgia (Bornholm disease)[4, 7, 9] and, in individual patients, infectious mono-

nucleosis, appendicitis, sinusitis, and influenza.[10] Different types of Coxsackie virus have been encountered in patients with herpangina[11] and in others with less distinctive but, at times, prevalent forms of acute self-limited disease.[7, 11, 12] In the cases cited below the accumulated evidence based on clinical laboratory and epidemiological findings appeared to favor the conclusion that the Coxsackie virus which was recovered from each patient was responsible for the associated disease. The incubation period of Coxsackie virus infection probably ranges from two to nine days.[7, 8, 11]

Illiness Resembling Nonparalytic Poliomyelitis

During the summer of 1948 among 157 patients with pleocytosis of the cerebrospinal fluid who were hospitalized in Connecticut and Rhode Island with a diagnosis of poliomyelitis or eseptic meningitis, none died and 113 (72%) were classified as nonparalytic. A significant discrepancy in the peak of incidence of nonparalytic and paralytic cases suggested that not all of the nonparalytic cases had poliomyelitis and that some of them represented a different disease entity.[4] Five patients from whom the Conn. -5 type virus was isolated came from three different parts of Connecticut. All were males, four were from five to 11 years and one was 32 years of age. The acute illness began suddenly in three, gradually in two, and had a diphasic course in two instances. All patients had fever which lasted from three to nine days. The maximal elevation of temperature ranged from 100.8 to 104.5° F. Headache, nausea, and abdominal pain were common initial complaints; stiffness of the neck or back and vomiting tended to develop later. Hyperemia of the pharynx was noted in two patients. None of the patients had abdominal tenderness, although four complained of abdominal pain. The total W.B.C. and differential counts in the blood were within the normal range. All had pleocytosis of the cerebrospinal fluid. The course of illness was relatively uncomplicated and terminated in complete recovery.

Epidemic Myalgia or Pleurodynia (Bornholm Disease)

This syndrome was first observed in 1856 by Finsen in Iceland. The illness is characterized by fever and severe pain located in various parts of the body particularly in the lower chest and abdomen. The onset is frequently abrupt, the course self-limited, and recovery is the rule. Evidence that Coxsackie viruses can induce Bornholm disease has been accumulating. A strain of Conn.-5 type virus appeared to be responsible for a sporadic case of pleurodynia hospitalized in New Haven, Connecticut, in 1948(4). Four of six laboratory workers who contracted Coxsackie virus infections (three by the Conn. -5 type and one by the Ohio-1 type) also had illnesses resembling Bornholm disease (7,8). Weller, Enders, Buckingham and Finn (9) tested specimens from representative patients among 114 admitted during August and September, 1947 to a hospital in Boston, Massachusetts with the diagnosis of epidemic pleurodynia. Strains of virus were recovered from the feces of four of these patients, and two were identified as Conn. -5 type virus. An increase in antibodies against this type of virus was detected in neutralization tests with specimens of serum from seven of the patients. The clinical features of illness in these twelve patients with pleurodynia and Coxsackie virus infection, were fever and pain in all, thoracic pain in 11, headache in eight, and abdominal pain in six. Four had stiffness of the neck or back. One of the laboratory workers had an abnormal number of leucocytes in the cerebrospinal fluid.

Herpangina and Other Forms of Minor Illness

Strains of type 2 virus were recovered from children in 1948 during outbreaks of "three day fever" described by Webb et al (12). Huebner et al (11) reported the recovery of Group A Coxsackie viruses of six different antigenic types from patients observed during the summer of 1950. These patients, mostly children, had brief febrile illnesses with mild sore throat and small vesicular or ulcerative lesions of the fauces. The fever usually subsided in three days, the ulcers shortly thereafter. Huebner and his associates pointed out the similarity of illness in these patients to a clinical entity "herpangina" described by Zahorsky (16).

Epidemiology

Coxsackie viruses have been recovered from individuals of both sexes and more frequently from children than from adults. Most isolations have been made in the summer and fall. Sufficient data is not yet available to establish the incidence according to sex and season nor to determine age specific attack rates. No reservoir of infection by these viruses other than man has been found. Present evidence suggests that the factors concerned with communicability in infection by Coxsackie viruses are comparable to those in poliomyelitis.

Treatment

No specific form of therapy is known which affects directly viruses of the Coxsackie group. At present, treatment is entirely supportive and symptomatic.

(Editorial note: references identified upon request)

Relationship of Pyelonephritis and Hypertension and its Prognostic Outlook

E. J. TANQUIST, M.D.
Alexandria, Minn.

Pyelonephritis refers to lesions in which both renal parenchyma and pelvis are involved. This is the most common disease of the kidney. Recently interest has centered about chronic pyelonephritis because it is thought to produce hypertension.

The literature is replete with articles upon the subject of bilateral and unilateral involvment of pyelonephritis. Of course the bilateral involvment of the kidneys do not offer any easy proof of the relationship of pyelonephritis and hypertension, but there are a large number of cases on record of unilateral pyelonephritis in which nephrectomy has resulted in a cure. Other renal diseases, such as tuberculosis, malformations and amyloid disease may be associated with hypertension and nephrectomy often brings the hypertension down to normal.

RELATIONSHIP OF HYPERTENSION TO KIDNEY DISEASE

Goldblatt's experiment demonstrating the effect of ischemia of kidney in producing hypertension has changed the conception of the relationship of the kidney to hypertension. Patients with sclerotic changes in the vascular system may or may not have hypertension, but if the

renal vessels are sufficiently involved in the process hypertension appears. The clinical course of hypertension is determined greatly by the progress of the vascular disease in the kidneys. Involvment of the kidney may be sufficient to cause chronic hypertension but does not progress rapidly enough to seriously impair renal excretory function, and consequently most hypertensive patients die of heart failure or of cerebral accidents before uremia, which means extensive involvment of kidneys, develops. Thus progress and extent of vascular disease in the kidneys determine whether essential hypertension runs a benign or a malignant course. Hypertension may be now regarded as a symptom of renal vascular disease.[25]

Essential hypertension, cause of which is not known, is a common disorder responsible for about one fourth of all deaths, after the age of fifty, in United States, thru its effect upon the cardiovascular renal system. The earliest manifestation of the disease is associated with arteriolar vasospasm. Resultant ischemia of the kidney is followed by further increase in the hypertension, probably thru the elaboration of some pressor substance in the kidney, and as a consequence of the impaired nutrition of the kidney. This peripheral resistance is not confined to the kidney but is observed rather generally throughout the body. Death in essential hypertension is frequently the result of coronary disease (50% of cases), or cerebral vascular accidents (25 to 35% of cases) and only 10% of cases due to renal failure.[22]

Volhard has said that in malignant hypertension the younger the patient and the higher the diastolic pressure, the greater the danger of malignant course, and such patients are much more likely to die of renal failure.

KIDNEY DISEASE AND HYPERTENSION

The kidneys play an important role in hypertension:

1) essential hypertension, eclampsia
2) Primary renal disease as—Glomerulonephritis, pyelonephritis, urinary obstruction and nephrosis malformation as polycystic renal disease and occlusion of renal vessels.[25]

Thus it is apparent that hypertension is of renal origin and appears as symptom of renal vascular disease associated with varying grades of arteriosclerosis in other parts of the body.[25]

More importance must be placed in the future on pyelitis and pyelonephritis of childhood and in pregnancy as a possible forerunner of chronic kidney disease and hypertension. This illness has been regarded too lightly. These conditions are serious and by frequent exacerbations and recurrences causes serious progressive renal damage.[22]

EARLY EVIDENCES OF HYPERTENSION

It is a well known fact that hypertension may exist for a long period without any symptom whatsoever. When the symptoms finally appear they may arise from organs bearing the brunt of the vascular disease process, as in event of heart disease or cerebral accident. The first time many individuals knew about the presence of hypertension was when examined for insurance, or perhaps during a general physical check up.

Langley makes the observation that young individuals with tendency to high normal blood pressure were found later to develop definite hypertension.[15] Hine and Lander of the Mayo clinic found, in examining a group of patients over a period of some ten years, that hypertensive disease was four or five times as frequent in patients who on first examination had high normal or mild degree of hypertension as those who

had normal on first examination.[15]

There is an increase incidence of hypertension in all patients with age. Incidence of hypertension increases with age of patient and parallel the incidence of marked renal vascular change[23]. Dr. Bell makes the astonishing observation that one half of the population over fifty years have some degree of hypertension and the great majority of which have moderate degree without symptoms.[3]

FAMILIAL TENDENCY OF HYPERTENSION

Langley states that the incidence of hypertension was five times as great in cases where there is familial history of the disease. Report in the Mayo papers for 1950 indicated a high familial incidence of hypertension, indicating that 76% of hypertensive patients gave a familial history of hypertension.[15]

PYELONEPHRITIS AND HYPERTENSION

Weiss and Parker 1939 estimate that pyelonephritis causes 20% of malignant hypertension and malignant hypertension is more apt to terminate in renal failure and have less incidence of cardiovascular complications than if of other origin.[12]

Pyelonephritis must be regarded as a forerunner of later renal failure with malignant hypertensive picture. The cases are numerous of pyelitis in children and those which accompany pregnancy which in later years develop serious condition. Smith et al report case of an 8 year old child and a 33 year old woman each with pyelonephritis of several years duration. At first the blood pressure was of moderate proportions but later each developed a typical malignant hypertensive picture and succumbed. They emphasize the cause and effect relationship of pyelitis and pyelonephritis and malignant phase of essential hypertension.[22]

The destructive stage of pylelonephritis is followed by constriction of the kidney, leading to renal ischemia and hypertension in many cases. Unilateral pyelonephritis with contracted kidney when removed may reduce the persistent hypertension.

Braasch and Jacobson found that under 50 years of age hypertension is twice as prevalent in bilateral pyelonephritis as in control group and Shure comes to much the same conclusion. They show that incidence of hypertension in chronic bilateral pyelonephritis parallel degree of damage to kidney as evidence by urogram, and state further that hypertension occurs twice as frequently in pyelonephritis where blood urea is elevated.[7-23]

Frequency of hypertension with atrophic pyelonephritis increases with degree of atrophy. Effect of nephrectomy on hypertension better in atrophic pyelonephritis than in an other unilateral renal disorder. Braasch feels 70% cases relieved by nephrectomy.[10-8] Dr. Braasch states further that surgical removal of unilateral lesion will often relieve hypertension. Such is the fact with chronic unilateral pyelonephritis which is the lesion that occurs most often with hypertension.[17]

Moderate hypertension may develop in uncomplicated bilateral pyelonephritis but severe prolonged hypertension is usually due to associated chronic glomerulonephritis or primary hypertension. Bell states further that it is difficult to see how unilateral pyelonephritis can cause hypertension when bilateral form of this disease seldom does. All renal disease with severe obstruction of capillaries, arteries or vein produce hypertension.[3] Some authors claim that chronic bilateral pyelonephritis contributes to incidence of hypertension, although hypertension found is of comparatively benign nature.[18]

Dr. Bell states that hypertension seldom occurs in uncomplicated chronic bilateral pyelonephritis and that a severe prolonged hypertension with cardiac hypertrophy is usually due to complicating lesion such as

chronic glomerulonephritis or renal atertiosclerosis.[3] Several writers have noted that bilateral pyelonephritis is occasionally accompanied by hypertension.

The interesting data on relation of unilateral renal disease to hypertension as presented by Pearman, et al suggest that pyelonephritis like adenomatous goiter and arthritis is merely asociated with hypertension and is not a causative factor.[3]

Braasch and Jacobson found in a study of 180 cases of chronic bilateral pyelonephritis an incidence of hypertension in 26% of cases and in a control group of 975 cases taken at random at Mayo Clinic there was an incidence of hypertension of 20%. Only a difference of 6%, which the different age groups might account for.[6-26] These authors found further that hypertension in 47% cases of chronic unilateral pyelonephritis and that hypertension associated with atrophic pyelonephritis will be relieved by nephrectomy in 70% cases.[8-26] Barker and Walters reported successful case of nephrectomy in chronic pyelonephritis and believes surgical removal of diseased kidney is indicated as treatment of hypertension in unilateral atrophic pyelonephritis.[2-26] Nesbit 1941 stated that most cases of unilateral chronic pyelonephritis with hypertension subjected to nephrectomy were cured or improved.[20-26]

PYELONEPHRITIS

Pyelonephritis is an inflammatory lesion of the interstitial tissues of the kidney with involvement of pelvis caused by lodgment of bacteria in the kidneys. Alexander reports finding healed pyelonephritis in nearly 15% of 1000 consecutive autopsies. The infection may be either of the ascending, by far the greatest in number, or of the hematogenous type. It is important to determine whether it is of the ascending or hematogenous route for on that solution hinges the success of the therapy.[9] Dr. Bell states that pyelonephritis is due to lodgment of bacteria in kidney tissue while glomerulonephritis is essentially due to bacteria toxins.[3]

Pathology

In simple acute pyelonephritis there are multiple foci of infection throughout interstitial tissue and the pelvis is reddened, slightly dilated and covered with inflammatory exudate. As process progresses glomeruli and tubules become obliterated by scar tissue and further progress results in more destruction of normal tissue. The severe form shows diffuse abscess formation throughout cortex with gangrene of renal pyramid. The recurrent type shows the presence of acute inflammation with areas of scars of healed pyelonephritis. Chronic pyelonephritis may be the end results of repeated acute attacks or may be of long standing low grade infection and the pathology is one of progressive destruction of renal tissue.

II Clinical Aspect

An acute attack of pyelonephritis is diagnosed by chills, fever, urgency and painful micturation, low back pain, tenderness in costo-vertebral angle and confirmed by finding pyuria and bacilluria. The course is usually of short duration. The more severe types are rare and usually fatal.[9]

In the recurrent type there is found frequent history of recurrent episodes of chills, fever, dysuria which suggests incomplete healing of initial attack or complete healing with recurrence. Regardless of which occurrence signified progression of disease and danger of chronicity and possibility of renal insufficiency is definitely increased with each attack.[9]

The chronic form is often difficult to diagnose. There is very little evidence of renal infection. Pallor, easy fatiguability, lassitude, dryness of skin, gradual loss of weight, anemia

out of proportion to illness, urea clearance, psp and rbc are low. Close questioning will reveal acute episodes. Bacilluria and pyuria only present intermittently but repeated cultures and microscopic examination of urine will reveal infection. Hematuria appears in majority of cases. Pylogram and laboratory findings clinches diagnosis. A typical case of chronic pyelonephritis shows a slowly progressive destruction of renal parenchyma by infection, and is either unrecognized or has been inadequately treated and thus never eradicated. B. Coli seems to be the principal source of infection.[9]

DIAGNOSIS

General physical examination, including study of size of heart, a carefully elicited history with special emphasis of repeated episodes of chills fever and urinary disturbances, observing condition of peripheral vessels, tests of kidney function, urinalysis, will enable one to establish a diagnosis in majority of cases.[22]

Then the presence of pus in urine calls for bacteriological study of the urine with study of renal function. Intravenous pyelogram and cystoscopic studies with retrograde pyelogram may be necessary. Such studies will reveal infectious agent, most frequently colon bacillus group, with evidence of obstructive lesion in urinary tract. Obstructive lesion is found repeatedly as cause of the trouble.[22]

Slow progress of pyelonephritis over a period of years may lead ultimately to renal insufficiency, frequently associated with intermittent or persistent hypertension. Case may be confused with glomerulonephritis, but urinary studies will reveal much pus, cells with few or no casts and bacterial study reveals organism usually of colon group and then we may feel certain we are dealing with pyelonephritis. The pyelogram will determine if obstructive lesion is responsible for infection. This differential diagnosis is important as pyelonephritis usually yields to adequate treatment by antibiotics and sulfonamides.[22]

Report of a personal case of Chronic Pyelonephritis with hypertension as of January 1950.[26]

Female, age 46, housewife. Seen first in 1944 for a minor pelvic ailment. Normal physical findings with normal blood pressure. Seen again in 1948 when she had a normal blood pressure and normal physical findings except with suffering with a large fibroid of uterus which was fixed in pelvis. Laboratory studies were normal.

A subtotal hysterectomy in Sept. 1948 revealed a large soft fibroid with massive attachments through marked inflammatory process to the pelvic wall, rectum and small bowels. Following this operation she developed hypertension, pressure rising up to 230/130, but patient was remarkably free from symptoms.

January 1949 an intravenous excretory pyelogram revealed a normal right kidney, pelvis and ureter and a functionless kidney on the left side. Cystoscopy revealed normal bladder and normal ureter, pelvis and kidney on the right side. Urine contained no pus or bacteria. However catheter could not be passed up the left ureter. PSP and ability to concentrate urine, blood urea were all normal. Bl P 230/130.

February 1949 she was subjected to a left nephrectomy and recovery was without incidence. Dr. Bell reported a severe chronic pyelonephritis. Blood pressure returned to normal quite promptly following the operation and the pressure has remained about 15/90, although occasionally the diastolic has registered 92 to 94. She was examined on April 20th 1951 and found in excellent health and pressure remaining essentially normal. In other words the pressure has remained normal for over two years.

This adds another successful case

of nephrectomy in unilateral pyelonephrtis with hypertension to the 20 successful cases reported by Bell.

DISCUSSION

There are a large number of cases of nephrectomy in unilateral renal disease with hypertension reported in the literature. The largest group of these renal disease is that of pyelonephritis. A study of the table presented by Dr. Bell in his excellent book on Renal Diseases and the excellent article by Dr. Langley brings out the fact that it is difficult to correctly evaluate the cures by nephrectomy of pyelonephritis and hypertension. This due to the fact that in many cases the record is inadequate and in others the required period of at least one year has not elapsed between the nephrectomy and the time of reporting cases.

Summary below of cases of unilateral renal diseases with hypertension treated by nephrectomy.

	Pyelonephritis Total: cases:	total cases	cured cases	% cured
Dr. Bell	61	26	18	
Dr. Ratliff	49	19	7	
Dr. Maitland	1	1	1	
	111	46	26	57%
Dr. Langley	103	35	20	57%

The 18 cases of Dr. Bells reported as cured, 6 should not, perhaps have been included because these were of less than a year after nephrectomy. Bell adds to his list of 61 cases those reported by Ratliff in 1947 out of a group of 2055 hypertensive cases. He reports 7 cases as cured. Then too one successful case of Maitland.[1] Omitting the 6 cases gives Bell success 20.

Langley analyzed 103 cases that he found in the literature of unilateral renal disease subjected to nephrectomy. Of that group he reported finding 47 as successful, and out of the 35 cases of pyelonephritis he found 20 successful cases. Langley believes nephrectomy is apt to be more successful in young patients with no familial history of hypertension; with the obvious finding of a non functioning kidney on the one side and a normal kidney on the other side. He believes unilateral renal disease is seldom cause of hypertension but when present occasionally blood pressure does return to normal after nephrectomy.

In discussing pyelonephritis the chronic form is of greatest importance and it offers the greatest diagnostic problem and is the form so important in diagnosis in order that proper treatment may be instituted. The acute form or acute recurrent form are less troublesome in diagnosis and should not be missed.

We have established thru the literature the fact that chronic bilateral pyelonephritis with hypertension is an incidental finding. However in unilateral renal disease we find that chronic pyelonephritis is an important finding as the authorities are agreed that nephrectomy offers the best treatment, in many, if not the majority of cases. Langley offers the best reason why there are failures in some of the cases. He concludes that in event of a definite case of chronic pyelonephritis, with at least moderate involvment, no familial incidence of hyperten-

sion and in event of the younger individuals nephrectomy should be successful in the majority of cases. Many individual reports confirm the same.

SUMMARY

Hypertension is not associated more often with chronic bilateral pyelonephritis than is found in a control group. Opinions vary but it is reasonable to conclude that bilateral pyelonephritis does not show as frequent appearance of hypertension as in the event of unilateral form. Some have concluded that bilateral pyelonephritis is an incidental finding as would be the case of arthritis or adenomatous goiter.

To diagnose pyelonephritis it is important to note, history of recurrent attacks of acute episodes, frequency, urinalysis, pyuria, bacilluria, culture, history of urinary obstruction and pyelogram. Diagnosis is important for early treatment usually means a cure.

It is important to diagnose pyelitis and pyelonephritis in children in order to properly handle the patient, as it is curable in most cases with antiobotics and sulfonamides. Recently it has been shown that one half of all women who have toxemia of pregnancy will develop hypertensive vascular disease or glomerulonephritis within a period of five years.

In the treatment of chronic pyelonephritis it is not sufficient to sterilize the urine but it is necessary that vigorous treatment be instituted by antiobotics and sulfonamides to insure an adequate concentration of the drug in the kidney tissue to eliminate the infectious process.

Chronic unilateral pyelonephritis with normal kidney on other side can often be successfully treated by nephrectomy, and percentage of cures varies but some report as high as 70% cures.

Prevention of hypertension is an important consideration. Obesity is an important etiological factor in hypertension. It is not so much a question of limiting meat and salt from the diet as the total diet which is important in regard to obesity. Tobacco seems definitely to be a vasospastic agent in some persons and should be denied to hypertensive and candidates for hypertension.

It is therefore of extreme importance to evaluate the history of past episodes of renal involvment, study of several specimens of urine, including microscopic, bacterial study and cultures of urine. It is also important to obtain history of familial hypertensive tendency and any history of long standing blood pressure rise or tendency to increased pressure.

(Editorial note: references identified upon request)

SIDE-GLANCES at HISTORY OF MEDICINE

BINOCULAR VISION

The basic fact of binocular direction localization, the law of identical visual directions, was first stated by Ewald Hering (Der Raumsinn und die Bewegungen des Auges, in: Handbuch der Physiologie by Ladmar Hermann, 1879, pt. I). Before Hering, however, P. L. Paum (Untersuchungen ueber Sehen mit zwei Augen. Kiel 1858; Ueber die einheitliche Verschmelzung verschiedenartiger Netzhauteindriecke beim Sehen mit zwei Augen. Arch. f. Anat. u. Physiol. 1861, pp. 63-111 and 178-227) already had pointed to the phenomenon of fusional areas; he showed that the images on the retina will, within certain limits, fuse even if the images do not fall exactly on corresponding points.

Conversion Hysteria

A visiting physician from Central America reported on two almost identical cases of psychogenic amblyopia. The first case was that of a 19 year old girl, reared in the very strict environment of a Spanish family, she was the only daughter. The father was a rigid domineering personality and the two older brothers displayed a rather condescending attitude toward the mother and more so toward the sister. The girl was engaged to a medical student. The family first had not approved of this engagement because the young man was partly of Indian descent but finally had given in although the student always was made aware of the fact that he was tolerated by the family.

The girl and the young man had lived in constant fear that the engagement in the last minute might be thwarted by a sudden interference on the part of the father or the brothers. The physician was called early one morning because the girl, on awakening had stated that she was blind. On examination she seemed not to be upset about the sudden loss of vision. The pupils reacted promptly to light and in accommodation. There was some blinking when the finger was brought close to the eye, yet less than would be expected in a person with normal vision. The fundus appeared normal throughout. The neurological examination did not reveal any abnormalities with exception of greatly increased tendon reflexes and a zone of anesthesia at the lower left arm which was independent of sensory nerve distribution. As the girl, as most people in Central America, had had several attacks of malaria in the past, the question of malarial amaurosis had to be considered which may develop rather suddenly in tertian malaria and which may progress to optical atrophy. Yet, the last malarial attack had happened one year ago and plasmodium vivax was not found in the blood. Other possible causes such as starvation amblyopia, tobacco amblyopia, alcohol or quinine amblyopia, amblyopia due to salicylic acid poisoning, bromism and mercurialism also could be dispensed with. As the girl was rather negativistic and refused to answer any questions dealing with emotional experiences, the physician resorted to interviewing the medical student. He admitted that the young girl and he had decided to have sexual intercourse in order to force a marriage when she would become pregnant. After there was no doubt about the pregnancy both had lost courage. And while the young man had avoided the house of his fiancee, she found a temporary solution in a flight into disease, "not willing to see reality." The situation was talked over with the family, and after the marriage date had been set the blindness cleared up within a few days.

A second case occurred several months later; the 24 year old unmarried daughter of a high official, studying in the United States, was called back because of her mother's serious illness. She expected to stay a short time at her home town and then to return to the United States. Her mother's unfavorable state of health, however, made it necessary to extend the length of her stay indefinitely. She was the only child. Her father was 25 years older than her mother, an irritable, unstable climateric man given to sprees of alcoholism and to outbreaks of violent temper. The mother was constantly on the defense which was

shrouded in immature mystic thinking projected into quack treatment of mentally ill persons. At the time, she was suffering from amebic dysentery. In this case, too, an almost total bilateral amblyopia developed within a period of three days. The neurological and opthalmoscopical examination was negative. Use of alcohol, tobacco, or drugs could be excluded. There was no malaria or virus infection in the previous history. An office interview disclosed that the young lady was in the third month of pregnancy. Because she was living in the United States, she had hoped her condition could be hidden from her parents. After she had been recalled, she had hoped to return to the United States within a short time. As her mother's condition would not permit this, she became frantic with fear considering her father's uncontrolled emotional reactions. Again the psychogenic dynamics symbolized to be "blind in order not to face reality." After a very difficult interview with the father and correspondence with the lover in the United States, it was decided to let her go back to the United States where the marriage would take place. After the solution of the problems involved the blindness subsided.

(Lit. A. N. Alling and O. A. Griffin. Diseases of the Eye and Ear. Lea & Bros. Philadelphia & New York, 1905, p. 8.—A. R. Hazelton. Nature of Starvation Amblyopia. J. Roy. Med. Corps. 86:171, April 1946—A. Fozzi and C. Iandolo. Malarial Amaurosis: Case of Malarial Optic Neuritis. Policlinico 55:481, April 19, 1948—D. O. Harrington. Ocular Manifestations of psychosomatic Disorders. J.A.M.A. 10:609, March 8, 1947).

BOOK REVIEWS

Books on Gynecology

A very instructive booklet for the expecting mother is Dr. Read's[1] lucid explanation of all the important questions a young mother, particularly a primipara may be confronted with. Pregnancy, labor, childbirth, care of the baby, hygienic problems, marriage relations, ante- and postnatal exercises are briefly but concisely treated. This small volume may prove to be a good help for the general practitioner in recommending it to his pregnant patients. Another book for lay-persons, but much larger in scope and purpose, is a medical guide[2] which is written with the aim to provide a book for the family physican which he can place in the hands of both mothers and daughters. In a very intelligent and interesting way the authors put before their female readers all questions which may trouble women, young and older, such as disorders of menstruation, backache, headache, constipation, fatigue, weight, cancer, marriage, pregnancy, childbirth, abortions, infertility. Although the style is plain, the presentation is never trite; the subjects are dealt with on the basis of broad experience and profound sympathy.

1. Introduction to Motherhood. By Grantly Dick Read, M.D. Harper & Brothers, Publishers, New York, 1950. 104 pages. Cloth $1.75.

2. The Modern Woman's Medical Guide edited by Aaron H. Horland, M.D. and Charles S. Steinberg, M.D. With a Foreword by R. B. Robins, M.D., Vice-President. American Medical Association. The World Publishing Company, Cleveland-New York, 1951. 393 pages. Cloth. $3.50.

Books on Internal Medicine

The 1951 edition of new and nonofficial remedies[1] is again a comprehensive, thorough, and painstakingly prepared reference book. The general practitioner will be mainly interested in chapters 4 and 5 (anti-infectives), 10 (blood formation and coagulation), 17 (hormones) and in Section C (unaccepted products). A very instructive presentation on bacteriological and laboratory work is Dr. Lord's[2] concise exposition. The essential procedures are briefly outlined in a succinct and generally understandable way. A very timely monograph deals with the effect of hormones on male sex organs[3] The physiology of these organs and the interaction of endocrine glands, the influence of hormonal groups on the male organs and problems such as hypo-and hypergonadism, castration syndromes, cryptorchidism, sterility, hormone treatment in prostatic cancer, sterility and impotence are dealt with lucidly. A topic with which the practitioner will become more and more conconcerned is decompression sickness.[4] The problems of high altitude and exposure to high pressure, particularly in high flights and exposure to explosives, are in the focus of modern medical problematics. The compilation by the National Research Council, therefore, should be studied carefully. A very useful guide to the structural understanding of malformed hearts is the late Dr. Spitzer's[5] paper of 1923 which has been well translated, analysed and summarized by Drs. Lev and Vass. It is an arresting and worthwhile reading. Briefly should be mentioned the volume XV of the Symposia on Quantitative Biology which has as subject ORIGIN and EVOLUTION of MAN. All aspects of the problem are displayed and scrutinized: genetics, historical biology, taxonomy, racial implications, constitution, adaptation and population questions.

1. New and Nonofficial Remedies, 1951. Issued under the direction of The Council on Pharmacy and Chemistry. Am. Med. Ass., J. B. Lippincott Company. 282 pages. Cloth. $3.
2. Determinative Bacteriology Manual. By Thomas H. Lord. Burgess Publishing Company. Minneapolis, Minn., 1951. 63 pages and 25 pages forms. Paper. $2.25.
3. The Effect of Hormones Upon The Testis and Accessory Sex Glands. by Morris J. Heckel, M.D. Charles C. Thomas, Publisher Springfield, Ill. 1951, 73 pages. Cloth. $2.5.
4. Decompression Sickness. Caisson Sickness, Diver's and Flier's Bends and Related Syndromes. Compiled under the Auspices of the Subcommittee on Decompression Sickness, Committee on Aviation Medicine, Division of Medical Sciences. National Research Council. Washington, D.C. W. B. Saunders Company, Philadelphia & London, 1951. 437 pages. Cloth. $8.50.
5. The Architecture of Normal and Malformed Hearts. A Phylogenetic Theory of Their Development. By Dr. Alexander Spitzer. Charles C. Thomas, Publisher. Springfield, Ill., 1951. 145 pages. Cloth. $5.
6. Cold Spring Harbor Symposia on Quantitative Biology. Vol. XV. Origin and Evolution of Man. The Biological Laboratory, Inc. Cold Spring Harbor, L. I., N.Y., 1951. 428 plus XII pages. Cloth. $7.

Books on Surgery

A very succint and informative on inhalation anesthesia[1] is Dr. Guedel's guide which now appears in its second edition. It introduces the reader into mechanisms and techniques and proceeds to the anesthetic accidents which are described briefly but comprehensively. A very useful book for the general practitioner. An extensive discussion on all aspects of liver injury[2] is presented in the Transactions of the 8th conference of the Macy Foundation. Particularly interesting for the practicing physician are the chapters on liver necrosis and on dietary

cirrhosis. A book on physical medicine which also deals with rehabilitation may be consistently mentioned in this section.[3] This textbook, edited by Dr. Krusen is a concise exposition of all methods of physical medicine; heat, massage, exercise, rest, ultraviolet radiation, diathermy, ultrasonic therapy, occupational therapy; it also shows forth diagnostic techniques which involve physical medicine such as electrodiagnostic, muscle testing, skin temperature measurements, etc. The section on rehabilitation is particularly important; it deals with rehabilitation of the spondylitic, arthritic, asthmatic hemiplegic, poliomyelitic, tuberculous, spastic, paraplegic patient (among others); the book concludes with essentials of functional anatomy. A very welcome presentation of a much discussed problem is Dr. Smithwick's compilation of surgery in hypertension. This short monograph contains an enumeration, description and evaluation of all recognized surgical precedures with statistical tables of results, with good illustrations and with a bibliography referring to the important publications in this field.

1. Inhalation Anesthesia. Second Edition by Arthur E. Guedel, M.D., The Macmillan Company, New York, 1951, 134 pages. Cloth $3.75.
2. Liver Injury. Transactions of the Eighth Conference, April 28 and 29, 1949, New York, N.Y., Josiah Macy, Jr., Foundation, New York, 1950, 164 pages, paper. $1.60.
3. Physical Medicine and Rehabilitation for the Clinician. Edited by Frank H. Krusen, M.D. W. B. Saunders Company, Philadelphia and London. 371 pages. Cloth, 1951. $6.50.
4. Surgical Measures in Hypertension. By Reginald, H. Smithwick, M.D. Charles C. Thomas, Publisher. Springfield, Illinois, 1951. 95 pages. Cloth. $3.

Books on Ophthalmology

A textbook of ophthalmology[1] which is suited for the general practitioner is the "concise treatise" prepared by Dr. Town and eleven contributors. The book starts rightly with methods of examination before considering the physiology of vision. This section is succinctly treated. It is followed by the description of disorders of vision and their correction. The next chapters deal with the external eye muscles and their abnormalities, with the orbit and its pathology, with the lacrimal apparatus and its diseases and with the eyelids. The following sections are concerned with conjunctiva, cornea, sclera, vitreous, uvea, lens, retina, and the optic nerve. The chapter on glaucoma and its treatment is very instructive. Very well prepared is the part on ophthalmic neurology. Ocular changes in systemic diseases, ocular therapeutics, and finally a splendid chapter on ophthalmic surgery conclude this welcome addition to the medical library. As an extension to the just mentioned book may be read Dr. Tait's volume on refraction.[2] This presentation describes in detail and comprehensiveness the pathophysiology of refraction errors, their diagnosis and testing, and the methods of refraction. All physicians who in their practice deal with ocular refraction will derive much benefit from this thoroughly conceived and clearly written book.

1. Ophthalmology. By Arno E. Town (Eleven Contributors), Lea & Febiger, Philadelphia, 1951. 511 pages. Cloth. $10.
2. Textbook of Refraction. By Edwin Forbes Tait, M.D. W. B. Saunders Company, Philadelphia and London. 1951. 371 pages. Cloth. $7.

Cancer of the Lip

About 16 to 25% of all primary carcinomata of the lower lip arise in premalignant lesions, such as hyperkeratosis or leukoplakia. Primary carcinoma of the lip occurs most frequently in the 6th and 7th decades. These primary cancers are observed more often in males than in females. C. C. Burkell (Canad. M.A.J., 62:28, January 1950) found 98.6% of these malignancies in males and 97% of the lesions were found on the lower lip. In author's series of 80 cases, 73 were males and 7 females. Hyperkeratosis or leukoplakia were present in 31; squamous cell carcinoma was diagnosed in 45, basal cell carcinoma in 4. The duration of the lesion was one year or less in 27, 1 to 3 years in 18, 17 years in one and 10 years in one. The author concludes that primary carcinoma of the lip should be curable in 100% and that irradiation is the treatment of choice; only in cervical lymph node metastasis is the prognosis poor inspite of radical dissection. (J. M. Neely. The Nebraska State Med. J., 8:225, August 1951).

Carcinoma of the Pancreas

In about 80 per cent of cases, the lesion develops without pain. Weakness and loss of appetite are the first symptoms. Jaundice and occasionally itching occur after a few days. An important characteristic in the diagnosis of cancer of the pancreas is the fact that when once jaundice has appeared and the stools have become alcholic, these signs do not recede. One of the most significant diagnostic features of carcinoma of the head of the pancreas is an enlarged, non-tender gallbladder which in the majority of the cases is palpable. (W. H. Cle. J. of the Michigan State Med. Soc. 50:481, May 1951).

Cisternal Puncture

The authors reviewed the reports of 44,771 cisternal punctures for diagnostic purposes within a five year period. Four deaths due to cisternal punctures were definitely established. The authors conclude that there is no sufficient justification for employing cisternal puncture, even by experienced physicians, either as a diagnostic means in syphilis of the central nervous system or as a follow-up procedure after treatment. (H. L. Cecil and E. B. Johnwick. J. of Vener. Dis. Inform., 4:86, April 1951).

Laryngeal Epilepsy (Tussive Syncope)

Laryngeal vertigo or laryngeal epilepsy is a syndrome in which severe coughing causes unconsciousness. Author contends the reason for the fainting may be seen in that, while coughing, the patient performs the Valsalva maneuvre of forced expiration against a closed glottis, thus increasing the intrathoracic pressure, causing venous congestion of the head, lowering the cardiac output, and bringing about cerebral anoxia. The author proposes to use the term—as introduced into the literature by W. S. McCann et al. (Arch. Int. Med., 84:845, 1949)—'tussic syncope' instead of the traditional designation laryngeal epilepsy or vertigo as the symptoms have no connection with the larynx, as vertigo is not a symptom, and as epilepsy occurs rarely and only as a complication in epileptic persons. When grand mal attacks are observed during the syncopal episode, it appears that the patient has a latent cerebral dysrhythmia (Lindsay E. Beaton. Arizona Med. 10:30, October 1951).

Nausea and Vomiting in Pregnancy

Nausea and vomiting occur in about 50% of women during the first three months of pregnancy. Author report on 96 patients requiring treatment for this syndrome. 66 patients were relieved by the routine treatment of small dry feeding. Of the 28 patients, not improved by this regimen, 9 were given pyridoxine intravenously with good results in 3 and with filure in 6. Five patients were given dramamine with failure in all cases. There were finally 25 patients not relieved by various measures tried. They, then, were placed on Resion (polyamine anion resin, synthetic sodium aluminum acetate and synthetic magnesium aluminum silicate — Medical Research Department, The National Drug Company, Philadelphia, Pa.). One to two tablespoons on rising, between meals and at bedtime was the dosage. 18 of the 25 patients responded favorably; 7 patients failed to improve. The authors felt that in these patients a psychogenic factor was responsible. (V. deP. Fitzpatrick. Am. J. Dig. Dis. 11:340. Nov. 1951)

Placenta Previa

Author is presenting a primarily report of three cases in which cystography confirmed the clinical diagnosis of placenta previa. The roentgen diagnosis was based essentially on the fixation and multiple constant defects seen on the superior border of the contrast filled urinary bladder, the asymmetry and irregularity of the bladder horns, especially seen in the oblique view, corresponding to the side of the low implanted placenta. (Mariano E. Garcia. Boletin del Colegio Medico Municipal de Sancti-Spritus, 17-18:18, January-February 1951)

New Penicillin Salt

"A new penicillin salt known as P-92 has been developed." This form of penicillin causes less reactions and also decreases severity of reaction in those in whom other forms of penicillin cause reactions. P-92 is a penicillin salt of N-methyl-1,2-diphenyl-2-hydroxyethylamine. It is prepared as a white powder and distributed in vials of 10 mm. capacity. Addition of 4.2 mm. distilled water makes a suspension containing 300,000 units. (New Orleans Med. & Surg. J. 4:13, Oct., 1951).

NEW PHARMACEUTICAL PRODUCTS

Efocaine. Nonoily depot solution for prolonged prevention of pain. According to the statement received, a single injection provides local anesthesia for as long as 6 to 12 days. The long duration of local anesthesia is founded on the fact that the anesthetic is slowly absorbed, as the anesthetic is deposited in crystalline form in an aqueous-miscible vehicle.

E. Fougera & Co., Inc., New York, N. Y.

Chloragel Tablets. Chlorophyll Fractions, Processed Ext., 25 mg; Aluminum Hydroxide Gel, Dried, 250 mg.; Magnesium Trisilicate, 250 mg.
Indications: gastric hyperacidity and peptic ulcer.

Walker Laboratories, Inc. (Formerly Walker Vitamin Products, Inc.), New York.

Desoxyn and Nembutal Capsules. Each capsule contains Desoxyn Hydrochloride (Methamphetamine Hydrochloride, Abbott), 5 mg., and Nembutal Sodium (Pentobarbital Sodium, Abbott), 30 mg.
Indications: emotional disturbances; nervous tension; fear; convalescence or prolonged illness complicated by depression; adjunct in management of obesity when patient is restless and tense.

Abbott Laboratories, North Chicago, Illinois.

SEND FOR THIS LITERATURE

1. **Allergies** (colds)—new antihistamine
2. **Dietary deficiencies**—daily vitamin supply
3. **Respiratory stimulant**—for the newborn
4. **Respiratory infections**—penicillin inhalation
5. **Psoriasis**—oral drug
6. **Bronchial asthma**—sustained management
7. **Cough**—antitussive syrup and tablets
8. **Sulfonamide** — triple sulfa combination
9. **Rheumatic fever**—management
10. **Antihistamines**
11. **Functional Uterine Bleeding** — management
12. **Pain control**—saddle block and higher spinal analgesia
13. **Whooping cough**—relief
14. **Premature labor** — habitual and threatened abortion—management
15. **Congestive heart failure**—mercurial diuretic
16. **Birth control**—diaphragm and jelly
17. **Sore throat**—alkaline germicidal solution
18. **Cough**—management
19. **Acute bronchitis**—treatment
20. **Inflammatory glandular swelling**—topical decongestive treatment
21. **Insomnia**—gentle sedation
22. **Appetite**—tonic
23. **Functional amenorrhea** — management
24. **Peptic ulcer**—demulcent
25. **Postoperative abdominal distention** and urinary retention — prevention and management
26. **Hemorrhoids**—relief of pain and inflammation
27. **Skin affections**—therapy
28. **Gastric neurosis**, pyloris spasm, nausea—management
29. **Sedative**—without habit formation
30. **Arthralgia** (bursitis), lumbago—ointment
31. **Pregnancy test**
32. **Allergies**—management
33. **Bronchial asthma**—prolonged relief
34. **Infant feeding**—milk-free formula in allergic reactions
35. **Infant nutrition**—information
36. **Cough**—palatable creosote preparation
37. **Eye hygiene**—decongestive lotion
38. **Headache**, neuralgia—pain relief
39. **Obesity**—dietary management
40. **Arthritis**—colloidal gold sulfide therapy
41. **Trichomoniasis**, moniliasis—vaginal suppositories
42. **Asthma**—symptomatic relief
43. **Pruritus**—management
44. **Cardiovascular disorders** — management
45. **Blood sugar**—12 minute test
46. **Urinalysis**—1 minute test
47. **Gout, Gouty Arthritis**—specific therapy
48. **Allergies** (food, inhalant, contact, etc.)—new parenteral therapy
49. **Dermatologic Conditions**—new parenteral therapy
50. **Ophthalmologic Conditions**—new parenteral therapy
51. **Acne and Pruritis Ani**—a cutaneous vasoconstrictor
52. **Arthritis**—a skeletal muscle relaxant
53. **Gastrointestinal and genitourinary spacticity**—a smooth muscle relaxant and sedative.
54. **Peripheral vascular disorder**—a vasodialator

Use Postage Free Card for Ordering Free Literature